PART 4

VITAL SIGNS

CHAPTER 15

Pulse Rate and Contour

KEY TEACHING POINTS

- Tachycardia (increased heart rate) portends a worse prognosis in many different conditions, including sepsis, pneumonia, myocardial infarction, acute gastrointestinal hemorrhage, gallstone pancreatitis, and stroke.
- The two most common abnormalities of pulse contour are pulsus alternans and pulsus paradoxus. Both are detectable by palpation or by using the blood pressure cuff.
- Pulsus alternans (regular rhythm with alternating strong and weak pulse) indicates severe left ventricular dysfunction.
- Pulsus paradoxus (inspiratory decline in systolic blood pressure of more than 10 to 12 mm Hg) appears in cardiac tamponade and severe asthma. In patients with significant pericardial effusions, the finding of pulsus paradoxus increases the probability that pericardiocentesis will improve cardiac output; its absence decreases the probability that pericardiocentesis will be beneficial.
- In patients with hypovolemic shock, the femoral pulse is the best indicator of cardiac perfusion.

PULSE RATE

I. INTRODUCTION

Taking the patient's pulse is one of the oldest physical examination techniques, practiced as long ago as 3500 BC by ancient Egyptian physicians, who believed a weakening pulse indicated advancing disease.[1] The pulse was one of Galen's (ca. 129–200 AD) favorite subjects, occupying several treatises that directed clinicians to observe the pulse's speed, force, and duration.[2,3] The first accurate observations of heart rate in disease were by John Foyer (1649–1734), who published his clinical observations in 1707 based on his invention, the pulse-watch.[3] The first clinicians to establish the significance of bradycardia were Adams and Stokes, who between 1827 and 1846 pointed out that not all seizures and fainting resulted from disease of the brain but instead could occur because of the slow pulse of heart block.[1]

II. TECHNIQUE

Most clinicians determine the pulse rate by palpating the radial pulse or, less often, by listening to heart tones with a stethoscope (i.e., apical rate). Counting the pulse for 30 seconds and doubling the result is more accurate than 15 seconds of observation.[4] In patients with fast heart rates, especially if the patient has atrial fibrillation, counting the apical rate is more accurate than counting the radial pulse, and 60 seconds of observation is more accurate than shorter periods.[5]

The difference between the radial pulse rate and the apical rate (the apical rate always being greater if there is a difference) is called the **pulse deficit**. A pulse deficit has traditionally been associated with atrial fibrillation, although it is a common finding with extrasystoles and all fast heart rates and by itself has little diagnostic significance.[6]

III. THE FINDING

Many textbooks state that the normal sinus rate ranges from 60 beats/minute to 100 beats/minute, but more recent information indicates that the heart rate of 95% of healthy persons instead ranges from 50 beats/minute to 95 beats/minute.[7] **Bradycardia** is a pulse rate less than 50 beats/minute; **tachycardia** is a rate greater than 100 beats/minute.

IV. CLINICAL SIGNIFICANCE

An important role of any vital sign is to provide the clinician with an early indication that trouble is afoot for the patient. EBM Box 15.1 shows that the finding of tachycardia serves this role well. In a wide variety of clinical disorders, including septic shock, pneumonia, myocardial infarction, upper gastrointestinal hemorrhage, gallstone pancreatitis, and pontine hemorrhage, the finding of tachycardia (variably defined as rate >90 beats/min to >110 beats/min) predicts both increased complications and decreased chances of survival (likelihood ratios [LRs] = 1.5 to 25.4). In patients with myocardial infarction, the increased risk of adverse outcome is a continuum, being greater for patients with higher heart rates and persisting whether or not the patient has a low ejection fraction, takes β-blocker medications, or receives thrombolytic therapy.[12,16-19] Tachycardia continues to predict increased mortality when detected during the first year after myocardial infarction.[20] In patients with septic shock, the relationship between tachycardia and mortality is independent of whether the patient receives vasopressor medications,[9] and in patients with pontine hemorrhage, tachycardia is a better predictor of mortality than other neurologic findings such as extensor posturing or the absence of withdrawal to pain.[15] The *absence* of tachycardia, on the other hand, decreases the probability of hospital mortality in patients with trauma, septic shock, and pontine hemorrhage (LRs = 0.1 to 0.3; see EBM Box 15.1) and argues *against* the presence of active bleeding during endoscopy for upper gastrointestinal hemorrhage (LR = 0.3).

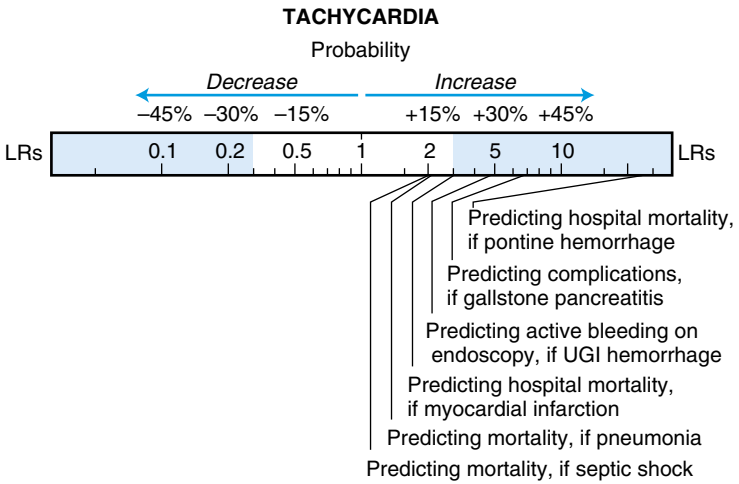
Bradycardia is also an ominous finding in acute disorders, particularly in patients presenting with severe trauma: in such patients, a pulse rate of 50 or less predicts mortality with a sensitivity of 17%, specificity of 99%, positive LR of 20.7, and negative LR of 0.8.[21]

Heart rates less than 50 beats/minute or greater than 120 beats/minute may also indicate heart rhythms other than sinus rhythm (e.g., complete heart block, atrial flutter), a subject discussed fully in Chapter 16.

EBM BOX 15.1
Tachycardia, Predicting Patient Outcome

Finding (Reference)	*Sensitivity (%)*	*Specificity (%)*	Likelihood Ratio* if Finding Is	
			Present	*Absent*
Heart Rate >90 beats/min				
Predicting hospital mortality, if trauma and hypotension[8]	94	38	1.5	**0.2**
Heart Rate >95 beats/min				
Predicting hospital mortality, if septic shock[9]	97	53	2.0	**0.1**
Heart Rate >100 beats/min				
Predicting mortality, if pneumonia[10]	45	78	2.1	NS
Predicting hospital mortality, if myocardial infarction[11,12]	6-9	97-98	**3.0**	NS
Predicting active bleeding on urgent endoscopy, if UGI hemorrhage[13]	71	86	**4.9**	**0.3**
Predicting complications, if gallstone pancreatitis[14]	86	87	**6.8**	NS
Heart rate >110 beats/min				
Predicting hospital mortality, if pontine hemorrhage[15]	70	97	**25.4**	**0.3**

*Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR.
NS, Not significant; *UGI,* upper gastrointestinal.
Click here to access calculator



ABNORMALITIES OF PULSE CONTOUR

I. PULSUS ALTERNANS

A. THE FINDING

Pulsus alternans describes a regular pulse that has alternating strong and weak beats (Fig. 15.1). The pulse must be absolutely regular to diagnose pulsus alternans and distinguish it from the bigeminal pulse, which also has beats of alternating strength, although the rhythm is irregular (see Chapter 16).[22] In rare cases of pulsus alternans, the weak pulse is so small it is imperceptible, with only half of the beats reaching the radial artery (**total alternans**).[23] Pulsus alternans is often accompanied by alternation of the intensity of heart sounds and murmurs (**auscultatory alternans**).[22,24] Traube first described pulsus alternans in 1872.[25]

B. TECHNIQUE

Palpating the radial pulse or using the blood pressure cuff is the best way to detect pulsus alternans. When using the blood pressure cuff, the clinician should stop deflating the cuff at the first appearance of Korotkoff sounds and hold the cuff pressure for several beats just below systolic blood pressure. In patients with pulsus alternans, only the Korotkoff sounds belonging to the strong beats are heard. Further deflation of the cuff allows cuff pressure to fall below the systolic pressure of the weaker beats, causing the cadence of Korotkoff sounds to suddenly double. The usual difference in systolic pressure between the strong and weak beats is only 15 to 20 mm Hg.[23]

Pulsus alternans often is most prominent in the several beats immediately after a pause in the heart rhythm. Typically, the pause is caused by a premature beat or the abrupt termination of a paroxysmal tachycardia.[26]

C. CLINICAL SIGNIFICANCE

In patients with normal heart rates, the finding of pulsus alternans indicates severe left ventricular dysfunction, caused by ischemic or valvular heart disease, long-standing hypertension, or idiopathic cardiomyopathy.[27-29] In one series of patients presenting for cardiac catheterization, investigators specifically looked for pulsus alternans after premature beats or 10 seconds of pacemaker-induced atrial tachycardia: those with pulsus alternans had worse ejection fractions and higher left ventricular filling pressures than those without the finding.[26]

In patients with rapid heart rates, pulsus alternans has less significance because even patients with normal hearts sometimes develop the finding during paroxysmal tachycardia.[30] Also, pulsus alternans rarely may reflect an intermittent left bundle branch block that alternates with ventricular beats having normal conduction.[31]

D. PATHOGENESIS

There has been considerable debate regarding whether the primary cause of pulsus alternans is alternation of intrinsic contractility of the heart (contractility argument) or alternation of filling of the ventricles (hemodynamic argument).

One version of the hemodynamic argument is particularly compelling.[25,32] In patients with a *regular* pulse, the sum of the length of systole and the length of the subsequent diastole must be constant. If systole lengthens for any reason, the subsequent diastole must be shorter; if systole shortens for any reason, the subsequent diastole must be longer. In patients with left ventricular dysfunction, a sudden increase in ventricular filling (such as that induced by a postextrasystolic pause) causes the subsequent systole to produce a strong beat, although it takes

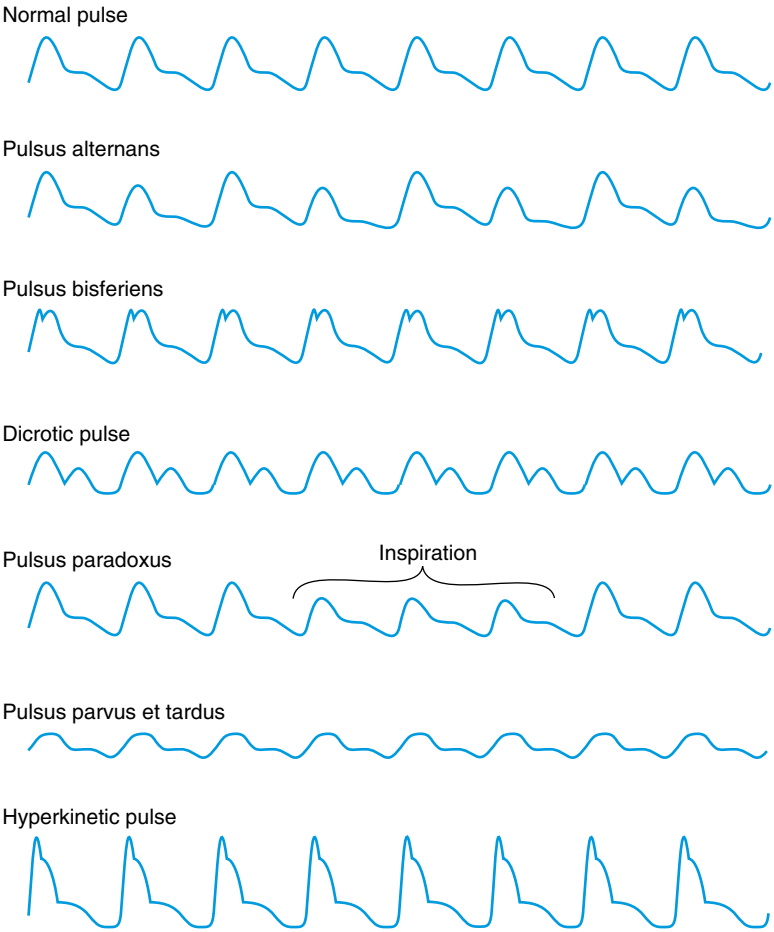


FIG. 15.1 ABNORMALITIES OF PULSE CONTOUR. The normal pulse tracing (*top row*) is displayed with six tracings of abnormal pulse contours (*bottom rows*). **Pulsus alternans** (*second row*) is a *regular* pulse that has alternating strong and weak beats. Both **pulsus bisferiens** (*third row*) and the **dicrotic pulse** (*fourth row*) have two beats per cardiac cycle: in pulsus bisferiens both beats are systolic, whereas in the dicrotic pulse one is systolic and the other diastolic. **Pulsus paradoxus** (*fifth row*) is a pulse whose systolic blood pressure falls more than 10 to 12 mm Hg during inspiration. **Pulsus parvus et tardus** (*sixth row*) is a pulse that has a small volume and rises slowly. The **hyperkinetic pulse** (*last row*) is a pulse with unusually abrupt and strong force; it may have a normal diastolic blood pressure (e.g., severe mitral insufficiency) or low diastolic blood pressure (e.g., severe aortic regurgitation). These tracings are facsimiles of actual pulse tracings made more than 100 years ago. See text for pathogenesis and clinical significance.

longer than normal for the weakened heart to eject this blood (i.e., thus lengthening systole). By prolonging systole, the strong beat thus shortens the next diastole, which reduces filling of the heart and causes the next beat to be weaker. The weaker beat is ejected more quickly, shortening systole and causing the next diastole to be longer, thus perpetuating the alternating pulse.

Nonetheless, the hemodynamic argument does not explain how pulsus alternans ever gets started when there is no pause in the rhythm from an extrasystole or termination of a tachycardia. Most experts now believe that alternation of intrinsic contractility is the fundamental problem in pulsus alternans, because alternation can even be demonstrated in vitro in isolated muscles at constant length and resting tension.[28,29] Once alternans begins, however, the hemodynamic effects probably contribute to the alternating amplitude of the pulse.

II. PULSUS BISFERIENS

A. THE FINDING

Pulsus bisferiens (Latin *bis*, meaning "twice," and Latin *ferire*, meaning "to beat") has two beats per cardiac cycle, both of which occur in systole (the first beat is called the **percussion wave**; the second, the **tidal wave**; see Fig. 15.1).[22] Descriptions of pulsus bisferiens appear in the writings of Galen.[33]

B. TECHNIQUE

Pulsus bisferiens is detected by palpating the brachial or carotid pulse with moderate compression of the vessel, or by using the blood pressure cuff.[34] When using the blood pressure cuff, the clinician hears a quick double tapping sound instead of the typical single sound. (The clinician can mimic the double sound by saying "pa-da…pa-da" as fast as possible.)[35]

C. CLINICAL SIGNIFICANCE

Pulsus bisferiens is a finding in patients with moderate-to-severe aortic regurgitation.[33,35,36] Pulsus bisferiens also occurs in patients with combined aortic stenosis and regurgitation, though the principal lesion is usually the regurgitation and the stenosis is mild.[33,36,37] There are exceptional cases of the finding in severe aortic stenosis.[34]

Pulsus bisferiens is sometimes described in patients with hypertrophic cardiomyopathy,[38] although almost always as a finding seen on direct intra-arterial pressure tracings, not as one palpated at the bedside.[39]

D. PATHOGENESIS

The bisferiens pulse probably results from rapid ejection of blood into a flexible aorta. Because of the Venturi effect, the rapidly moving bloodstream temporarily draws the walls of the aorta together, reducing flow momentarily and producing a notch with two systolic peaks in the waveform. (In hypertrophic cardiomyopathy, the Venturi effect draws the anterior leaflet of the mitral valve and the interventricular septum together.)[34,40] Although this hypothesis was proposed over 50 years ago, direct evidence supporting it is difficult to find.

III. PULSUS PARADOXUS

A. THE FINDING

Pulsus paradoxus is an exaggerated decrease of systolic blood pressure during inspiration (see Fig. 15.1).[22,41] Although the usual definition is an inspiratory fall in systolic blood pressure exceeding 10 mm Hg, a better threshold may be 12 mm Hg, which is the upper 95% confidence interval for inspiratory decline in normal persons (i.e., the average inspiratory decrease in systolic pressure of normal persons

is 6 ± 3 mm Hg).[42] In patients with pulsus paradoxus, the systolic blood pressure and pulse pressure fall dramatically during inspiration, though the diastolic blood pressure changes little.[41,42]

In 1873, Kussmaul first described pulsus paradoxus in three patients with pericardial disease.[43,44] Kussmaul called the finding "paradoxical" because the pulse of his patients disappeared during inspiration even though the apical beat persisted throughout the respiratory cycle. The term is unfortunate, because the finding is nothing more than an exaggeration of normal physiologic change.

B. TECHNIQUE

When checking for pulsus paradoxus, the clinician should have the patient breathe quietly and regularly, because even normal persons can induce a pulsus paradoxus with vigorous respirations. Pulsus paradoxus is detected by palpating the pulse or using the blood pressure cuff, although only paradoxical pulses exceeding 15 to 20 mm Hg are palpable.[45,46] For this reason, most clinicians use the blood pressure cuff, which has the added advantage of quantifying the finding (Fig. 15.2).

Pulsus paradoxus also has been noted in pulse oximetry tracings as respiratory movement of the tracing's baseline.[47] The amplitude of this oscillation correlates with the severity of pulsus paradoxus.[47] When using the blood pressure cuff to quantify pulsus paradoxus, clinicians may actually look at the visual display of the pulse oximeter instead of listening to the Korotkoff sounds.[48]

C. CLINICAL SIGNIFICANCE

Pulsus paradoxus is a common finding in two conditions: cardiac tamponade and acute asthma.

1. CARDIAC TAMPONADE

Pulsus paradoxus of more than 10 mm Hg occurs in 98% of patients with cardiac tamponade (i.e., a pericardial effusion under high pressure compressing the heart and compromising cardiac output; see Chapter 47). Because it is one of three key findings of tamponade—the others being elevated neck veins (sensitivity = 100%) and tachycardia (sensitivity = 81% to 100%)—the clinician should consider tamponade and check for pulsus paradoxus in any patient suspected of having pericardial disease, such as those with elevated neck veins, unexplained dyspnea, pericardial rub, or known pericardial effusion.[46]

In patients with pericardial effusions, the finding of pulsus paradoxus of more than 12 mm Hg discriminates patients with tamponade from those without tamponade, with a sensitivity of 98%, specificity of 83%, positive LR of 5.9, and negative LR of 0.03.*,[42]

2. CARDIAC TAMPONADE WITHOUT PULSUS PARADOXUS

In only 2% of patients with tamponade, pulsus paradoxus is absent. These patients usually have one of five disorders: (1) atrial septal defect, (2) severe left ventricular dysfunction (especially those with uremic pericarditis),[49] (3) regional tamponade (tamponade affecting only one or two heart chambers, a complication of cardiac surgery),[50] (4) severe hypotension,[51-53] or (5) aortic regurgitation. Knowing that aortic regurgitation may eliminate pulsus paradoxus is especially significant, because patients with proximal (type A) aortic dissection and hemopericardium usually lack the paradoxical pulse despite significant tamponade, and the unaware clinician may exclude the possibility of tamponade to the harm of the patient.

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*Tamponade was defined in this study as improvement in cardiac output of 20% or more following pericardiocentesis (see Chapter 47).

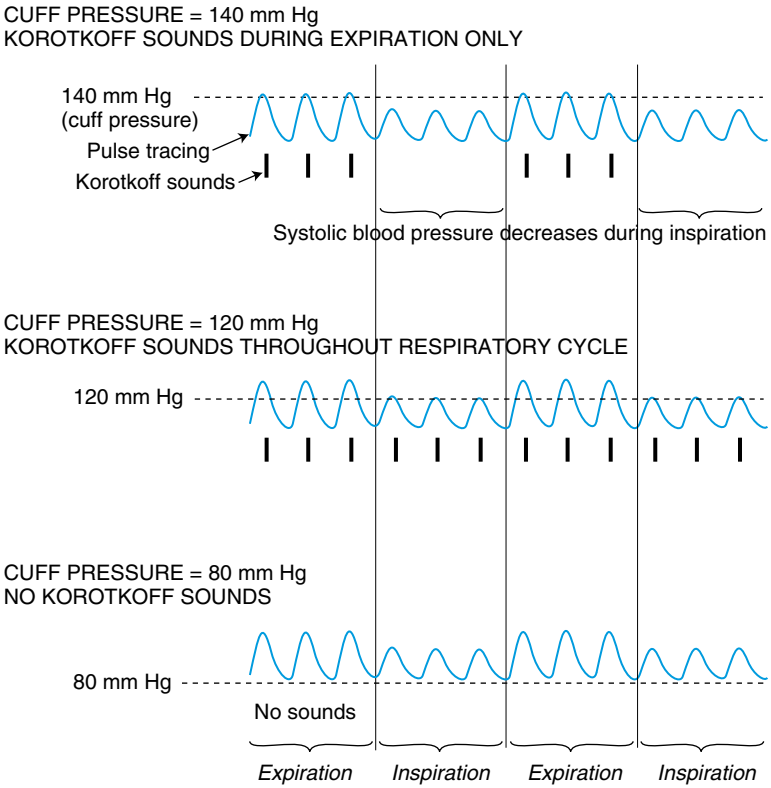


FIG. 15.2 TECHNIQUE FOR MEASURING PULSUS PARADOXUS. The figure simultaneously depicts the pressure in the blood pressure cuff (*dashed horizontal line*), the patient's pulse tracing (*solid line*), and Korotkoff sounds (*solid vertical bars* under pulse tracing) during two breaths (expiration and inspiration are separated by *vertical lines*). The pulse tracing shows the fall in systolic pressure during inspiration, which is characteristic of pulsus paradoxus. To detect and measure the paradoxical pulse, the clinician begins by checking the blood pressure in the usual way but slowly deflates the cuff to precisely identify the *cuff pressure* at three points: First, the moment Korotkoff sounds first appear (*top tracing*). In patients with pulsus paradoxus, cuff pressure will fall below the systolic pressure of just the expiratory beats, and the Korotkoff sounds will repeatedly come and go during quiet respiration, disappearing with inspiration and reappearing with expiration. Second, the moment when Korotkoff sounds persist throughout the respiratory cycle (*middle tracing*). At this point, cuff pressure has fallen below systolic blood pressure of all beats. Third, the moment when Korotkoff sounds disappear (i.e., the diastolic pressure, *bottom tracing*). In this patient, only expiratory Korotkoff sounds are heard between cuff pressures of 140 mm Hg and 120 mm Hg, but Korotkoff sounds are heard throughout the respiratory cycle between pressures of 120 mm Hg and 80 mm Hg. The patient's blood pressure is therefore "140/80 mm Hg with a paradox of 20 mm Hg" (i.e., 20 = 140 – 120).

The section on pathogenesis explains why pulsus paradoxus is absent in these clinical disorders.

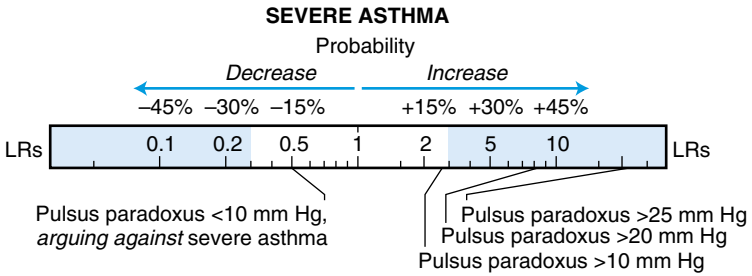
3. ASTHMA

EBM Box 15.2 shows that in patients with acute asthma, pulsus paradoxus exceeding 20 mm Hg almost certainly indicates severe bronchospasm (LR = 8.2).

EBM BOX 15.2
*Pulsus Paradoxus Predicting Severe Asthma**

Finding (Reference)	Sensitivity (%)	Specificity (%)	Likelihood Ratio† if Finding Is	
			Present	Absent
Pulsus paradoxus >10 mm Hg [45,54-56]	52-68	69-92	2.7	0.5
Pulsus paradoxus >20 mm Hg [45,54,55]	19-39	91-100	**8.2**	0.8
Pulsus paradoxus >25 mm Hg[56]	16	99	**22.6**	0.8

*Diagnostic standard: for *severe asthma,* a FEV1/FVC <50%,[45] FEV1 <1.0 L,[54] peak flow <200 L/min,[56] and peak flow <30% predicted.[55] All patients in these studies had acute asthma.
†Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR.
FEV_1, Forced expiratory volume in 1 second; *FVC,* forced vital capacity.
Click here to access calculator



Nonetheless, pulsus paradoxus has limited clinical utility in patients with acute asthma for two reasons: First, up to half of patients with severe bronchospasm lack a pulsus paradoxus greater than 10 mm Hg (see EBM Box 15.2). The sensitivity is low because in asthma pulsus paradoxus depends on both respiratory rate and effort, even when the degree of airway obstruction remains constant,[55,57] Second, the best measure of bronchospasm (and the criterion standard in EBM Box 15.2) is peak expiratory flow rate. In a busy emergency department with an anxious and dyspneic patient, it is much more convenient to measure peak flow rates using handheld flow meters than trying to interpret the coming and going of Korotkoff sounds.

In patients being mechanically ventilated, the amount of pulsus paradoxus, as reflected in the changing baseline of the pulse oximeter tracing, correlates with the degree of the patient's auto-PEEP (a measure of expiratory obstruction in ventilated patients).[47]

4. PULSUS PARADOXUS IN OTHER CONDITIONS

Pulsus paradoxus has been described in constrictive pericarditis, right ventricular infarction, pulmonary embolism, tension left hydrothorax, and severe pectus

excavatum,[41,58,59] although in each of these disorders it is an uncommon finding (see Chapter 47).

5. REVERSED PULSUS PARADOXUS[60]

Reversed pulsus paradoxus is a systolic blood pressure that falls more than 10 mm Hg *during expiration*. It has been described in three clinical disorders: (1) hypertrophic cardiomyopathy; (2) isorhythmic dissociation (i.e., inspiration accelerates the sinus rate, which temporarily positions the P waves before the QRS complex, thus coordinating the atrial and ventricular contractions and raising blood pressure; expiration slows the sinus rate, removes atrioventricular coordination, and lowers blood pressure); and (3) intermittent inspiratory positive-pressure breathing in the presence of left ventricular failure (this is a variation of the Valsalva square wave response in heart failure; see Chapter 48).

D. PATHOGENESIS

1. CARDIAC TAMPONADE

Tamponade develops when the pressure of fluid inside the pericardial space exceeds the diastolic filling pressure of the heart chambers. Once this occurs, the diastolic pressure in the heart chambers, reflected in the neck veins, becomes a measurement of the force acting to compress the heart. The four chambers, now smaller in size, begin to compete for space, and an increase in the size of one comes at the expense of the size of another. Inspiration increases, filling to the right side of the heart, and shifts the interventricular septum to the left and posteriorly, thus obliterating the left ventricular chamber and causing the cardiac output to fall. During expiration, the filling of the right side of the heart is less, which increases left ventricular size, and both cardiac output and blood pressure increase.[41,50,61-64]

This explains why pulsus paradoxus is absent in regional tamponade and tamponade associated with atrial septal defect, severe left ventricular dysfunction, and aortic insufficiency (see the section on Cardiac Tamponade Without Pulsus Paradoxus). Inspiratory movement of the interventricular septum is prevented when the right ventricle does not fill more during inspiration (atrial septal defect; see Chapter 40), when the left ventricular pressures are very high (severe left ventricular dysfunction), or when the left ventricle fills from some source other than the left atrium (aortic insufficiency). Regional tamponade, by definition, compresses only one or two chambers, enough to impair cardiac output but too confined to cause the heart chambers to compete for space.

2. ASTHMA

The mechanism of pulsus paradoxus in asthma is complex and not fully understood. Difficulty breathing causes wide swings of intrapleural pressure, which then are transmitted directly to the aorta, contributing to the paradoxical pulse. This is not a complete explanation, however, because the amount of pulsus paradoxus in asthma often exceeds the pressure shifts of these respiratory excursions.[57] Furthermore, the pulse pressure also declines during inspiration of some asthma patients, which would not happen if transmission of pressures were the only cause. Other proposed mechanisms are an inspiratory reduction in pulmonary venous return to the left heart[41,57,65,66] and the compressive action of the hyperinflated chest, which, like tamponade, may reduce the size of the heart chambers and cause them to compete for space.[55,67]

IV. PULSUS PARVUS ET TARDUS

A. THE FINDING AND TECHNIQUE

Pulsus parvus et tardus describes a carotid pulse with a small volume (pulsus parvus) that rises slowly and has a delayed systolic peak (pulsus tardus; see Fig. 15.1).[22] It is routinely detected by palpation.

B. CLINICAL SIGNIFICANCE

Pulsus parvus et tardus is a finding of aortic stenosis. Of its two components, pulsus tardus is the better discriminator, detecting severe aortic stenosis with a sensitivity of 31% to 91%, specificity of 68% to 93%, positive LR of 3.5, and negative LR of 0.4 (see Chapter 44).

C. PATHOGENESIS

Pulsus tardus depends on both obstruction to flow and the compliance of the vessel distal to the obstruction. The pulse waveform rises rapidly in stiff vessels but slowly in more compliant vessels that act like low-pass filters and remove the high frequency components of the waveform.[68] That the delay in the pulse reflects the severity of obstruction is a principle also used by Doppler sonography to gauge the severity of renal artery stenosis.[68]

V. DICROTIC PULSE

A. THE FINDING AND TECHNIQUE

The dicrotic pulse has two beats per cardiac cycle, but unlike pulsus bisferiens, one peak is systolic and the other is diastolic (see Fig. 15.1).[22] It is usually detected by palpation of the carotid artery.[69]

The second wave of the dicrotic pulse is identical in timing to the small dicrotic wave of normal persons, obvious on arterial pressure tracings but never palpable. The dicrotic wave is felt to represent the rebound of blood against the closed aortic valve.

B. CLINICAL SIGNIFICANCE

The dicrotic pulse occurs in younger patients with severe myocardial dysfunction, low stroke volumes, and high systemic resistance.[69,70] In patients who have had valvular replacement surgery, the finding of a persistent dicrotic pulse is associated with a poor prognosis.[70]

C. PATHOGENESIS

A dicrotic pulse relies on the simultaneous presence of two conditions: (1) low stroke volume, which significantly lowers the height of the pulse's initial systolic wave, thus increasing the chances that the dicrotic wave will be palpable;[71] and (2) a resilient arterial system, which amplifies the rebound of the pulse waveform during diastole. The importance of a resilient arterial system may explain why the dicrotic pulse usually occurs in young patients with cardiomyopathy, who have more compliant vessels than older patients.[69,70]

The importance of a low stroke volume to the dicrotic pulse is illustrated by the observation that the dicrotic pulse sometimes disappears with beats that have larger stroke volumes, such as the beat after a premature beat, the stronger beats of pulsus alternans,

and the expiratory beats of pulsus paradoxus.[69,71] Vasodilators often cause the dicrotic pulse to disappear, perhaps because of better forward flow and a greater stroke volume.[69]

VI. HYPERKINETIC PULSE

A. THE FINDING

The hyperkinetic pulse strikes the examiner's fingers with unusually abrupt and strong force (see Fig. 15.1). Hyperkinetic pulses may have either a normal pulse pressure (e.g., severe mitral regurgitation, hypertrophic obstructive cardiomyopathy) or increased pulse pressure (e.g., aortic insufficiency and other disorders with abnormal aortic runoff).[22] In both severe mitral regurgitation and hypertrophic obstructive cardiomyopathy, the blood is ejected rapidly from the left ventricle but the integrity of the aortic valve preserves a normal arterial diastolic and pulse pressure.[72] In aortic regurgitation, the rapid ejection of blood is accompanied by an incompetent aortic valve, which causes a very low diastolic pressure in the aortic root, thus increasing the pulse pressure and producing the Corrigan or water hammer pulse characteristic of this disorder (see Chapter 45).

B. CLINICAL SIGNIFICANCE

Chapter 45 discusses the significance of the water hammer pulse and large pulse pressure of aortic regurgitation.

In patients with mitral stenosis, the pulse is characteristically normal or diminished. If the clinician instead finds a hyperkinetic pulse in these patients, the probability is high that additional valvular disease is present, such as significant mitral regurgitation (sensitivity 71%, specificity 95%, positive LR = 14.2, negative LR = 0.3; see Chapter 46).[73]

VII. PULSES AND HYPOVOLEMIC SHOCK

In patients with hypovolemic shock, the peripheral pulses provide a rough guide to the patient's systolic blood pressure.[74] As blood pressure progressively diminishes, the radial pulse generally disappears first, then the femoral pulse, and finally the carotid pulse. In one study of 20 patients with hypovolemic shock, summarized in EBM Box 15.3, the femoral pulse had the greatest diagnostic accuracy in determining severity of shock: the presence of a palpable femoral pulse increased the probability of a systolic blood pressure greater than 60 mm Hg (LR = 2.9), whereas its absence decreased the probability of a blood pressure this high (LR = 0.1).

EBM BOX 15.3
Pulses and Hypovolemic Shock

Finding (Reference)	Sensitivity (%)	Specificity (%)	Likelihood Ratio† if Finding Is	
			Present	Absent
Detecting Systolic Blood Pressure ≥60 mm Hg**,74				
Carotid pulse present	95	22	NS	NS
Femoral pulse present	95	67	2.9	**0.1**
Radial pulse present	52	89	NS	0.5

*Diagnostic standard: for *systolic blood pressure*, invasive arterial blood pressure measurements.
†Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR.
NS, Not significant.
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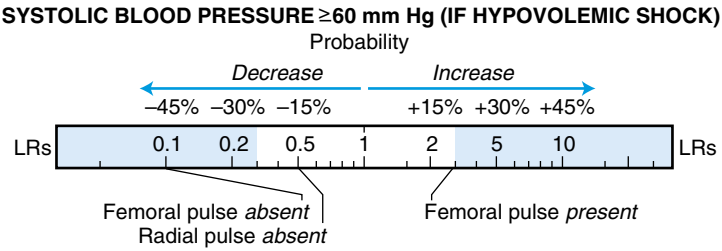

The references for this chapter can be found on www.expertconsult.com.

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